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UNIVERSITY OF ALBERTA

UPPER EXTREMITY PERIPHERAL NERVE ENTRAPMENTS AMONG WHEELCHAIR ATHLETES

BY

ROBERT BURNHAM

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Science.

DEPARTMENT OF PHYSICAL EDUCATION AND SPORT STUDIES

Edmonton, Alberta

Fall, 1993

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ISBN 0-315-88153-4

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DEGREE: Master of Science

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The undersigned certify that they have read and recommend to the Faculty of Graduate Studies and Research for acceptance, a masters thesis entitled "Upper Extremity Peripheral Nerve Entrapments Among Wheelchair Athletes" submitted by Robert Burnham in partial fulfillment of the requirements for the degree of Master of Science.

Dr. Robert Steadward

Dr. David Reid

Dr. H. Arthur Quinney

Dr. Brian Fisher

DEDICATION

This work is dedicated to my father, Dr. K.A. Burnham who, by example, instilled a desire to question, analyze and hypothesize - "QUAECUMQUE VERA"

ABSTRACT

Wheelchair sport is becoming increasingly popular and competitive. Paralleling this is the occurrence of sport related injuries and the need for quality sport medicine care for these unique athletes. Existing literature profiling wheelchair athletes' injuries has identified hand injuries, including symptoms of numbness, pain and weakness, suggesting nerve entrapment to be common. The purpose of this thesis was to conduct a series of investigations pertaining to upper extremity peripheral nerve entrapments among wheelchair athletes to identify: a) their prevalence, b) location, c) demographic and training factors associated with upper extremity peripheral nerve entrapment occurrence, d) a model of acute wheeling related nerve dysfunction, and e) whether glove protection of the hand would minimize wheeling induced nerve dysfunction.

To study purposes a, b, and c, clinical assessment and nerve conduction studies of the median, ulnar and radial nerves (including short segment stimulations across the carpal tunnel and ulnar groove) were performed on both arms of 28 wheelchair athletes and 30 able-bodied healthy controls. According to the clinical and electrodiagnostic (EDX) criteria established for nerve entrapment (> 2 SD from control means), UEPNE prevalence among wheelchair athletes was 23% clinically and 61% electrodiagnostically. Median nerve entrapment at the carpal tunnel was the most common site (46% prevalence) and the proximal portion of the carpal tunnel was most severely affected. Ulnar neuropathy at the wrist/hand (39% prevalence) and forearm were also common. Impaired median nerve function was found to be associated with disability duration, but not to other demographic or training/sport related factors.

To study purposes d and e, pre and immediately post-exercise EDX testing of the median nerve across the carpal tunnel was performed on both arms of 35 wheeling (hand trauma) and 11 cycling (no hand trauma) subjects. Each subject in the wheeling group wore a glove on one hand which had added padding over the carpal tunnel. A significant increase in across carpal tunnel conduction block (reflecting nerve dysfunction) was measured in the wheeling group but not in the cycling group. However, the amount of conduction block was not altered by glove use.

ACKNOWLEDGEMENTS

The author expresses sincere appreciation to a few of many individuals who contributed to his graduate experience and particularly the completion of this work:

Dr. Robert Steadward for his guidance and support of this project and his vision of the role of fitness, sport and sport medicine among athletes with a disability.

Dr. David Reid for his long time association and mentorship in sport medicine, concern for individuals with physical disabilities, and guidance as a member of the supervisory committee.

Dr. H. Arthur Quinney for his suggestions as a member of the supervisory committee.

Dr. Brian Fisher for his suggestions as a member of the supervisory committee.

Dr. Dorothy J. Carroll for her expert input regarding the electrophysiological basis of this project.

Joanne McAdam for her secretarial and editorial assistance.

The Sport Medicine Council of Alberta for financial assistance.

The cooperation and inspiration of the **athletes** who participated in this research.

My dear wife, **Colette** and children **Christie, Joseph, Scott, Taylor, Matthew, Nicole** and **Celeste**, for their ongoing and tireless support and for helping me keep balance and perspective in my life.

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LIST OF SYMBOLS AND ABBREVIATIONS

°C	degrees Celcius
C8/T1	eighth cervical and first thoracic spinal level
cm	centimetres
CMAP	compound motor action potential
CTS	carpal tunnel syndrome
CV	conduction velocity
DIP joint	distal interphalangeal joint
EDX	electrodiagnostic
Hz	Hertz - cycles per second
IDI	first dorsal interosseous
KHz	kilohertz
M.-E.-O line	a line intersecting the medial epicondyle and olecranon with the elbow flexed to 90 degrees
mmHg	millimetres of mercury
m/s	metres per second
ms	milliseconds
mV	millivolt
μV	microvolt
n	number
p	probability of type 1 error; level of significance
PIP joint	proximal interphalangeal joint
SD	standard deviation
SNAP	compound sensory action potential

CHAPTER 1.
INTRODUCTION

Sport for athletes with a disability was first introduced by Sir Ludwig Guttmann in 1948. At that time it was a means of providing therapeutic exercise for the spinal cord injured war veterans at the Spinal Injury Centre of the Stoke Mandeville Hospital in England. By 1952, annual international competitions were established and grew rapidly in popularity. In 1960, the first Paralympic Games were held at the site of, and near the same time as the able-bodied Olympics. Thereafter, other disability groups became interested in sport, and in 1976 amputee and blind athletes joined the spinal injured competitors at Toronto for the "1976 Olympiad for the Physically Disabled". In 1980, cerebral palsy athletes became a participating disability group in the Paralympic Games.[1] Sport for individuals with physical disabilities has gained rapid and relatively recent popularity. The 1948 competitions organized by Sir Ludwig Guttman involved only 16 competitors, whereas the 1992 Summer Paralympic Games involved 3032 competitors from 96 countries around the world. In Canada, participation in the Canadian Wheelchair Basketball League doubled between 1986 and 1991.[2]

Concurrent with the increasing participation of athletes with physical disabilities in wheelchair sport, is their level of competition and athletic excellence. Previously held records are regularly broken by large margins, which may in part be due to increased intensity and sophistication of training, availability of adapted training facilities, improved coaching, and advances in sport equipment design. No longer do wheelchair athletes consider sport simply as part of their rehabilitation. For many it is an important and healthy part of their recreational lifestyle. For others, preparing for and participating in high level national and international competitions is their full-time occupation, and considerable financial rewards and sponsorships are dependent upon their performance. Wheelchair athletes are increasingly looking to sport scientists for better ways to train, and to the medical/paramedical professions for improved techniques of injury prevention and treatment.[3]

Unfortunately, sport medicine for individuals with physical disabilities is in its infancy. Most of the research to date has been epidemiologic in terms of case reports, case series and cross-sectional surveys. Currently, injury profiles for various types of wheelchair sport and disability

groups are still being identified. Randomized control intervention studies are non-existent in the current wheelchair sport medicine literature.

Although limited, the existing wheelchair sport medicine epidemiologic research has described a relatively consistent profile of injuries sustained by wheelchair athletes. Upper extremity injuries predominate. The type and frequency of upper extremity injuries depends on injury definition. When the definition is left up to the athlete, skin and soft tissue injuries of the hands tend to be most prevalent.[2,4,5] However, when the injury definition is based on time lost from sport, the shoulder becomes the most prevalent site.[6,7] Shoulder problems have also been documented to require the greatest amount of medical/paramedical care.[2] The most common clinical diagnosis of these shoulder problems is tendinitis/bursitis (rotator cuff impingement syndrome).[3] In addition to hand skin and soft tissue injuries commonly reported by wheelchair athletes, symptoms of hand numbness, tingling and weakness also are common.

Obviously much more research needs to be done in order to provide for these athletes, who are involved in unique sports using unique equipment with unique physiologic responses to exercise, the type of sport medicine care that is currently available for able bodied athletes. Research priorities need to be set and should focus on:

a. injuries that are the most common (i.e. hand injuries),
b. injuries that are the most troublesome (i.e. shoulder injuries), and
c. injuries or conditions that are potentially the most dangerous (i.e. autonomic dysreflexia)
d. injuries that can be investigated with current technology (i.e. electrophysiological testing for peripheral nerve entrapment).

This thesis has chosen to look at the issue of peripheral nerve entrapments of the upper extremities of wheelchair athletes. The rationale for doing so is that the epidemiologic research would suggest that hand injuries are common, and hand numbness/weakness/pain suggestive of nerve entrapment, makes up part of the reported hand complaints. Additionally, peripheral nerve entrapment could result in additional neurologic impairment and may be treatable or even preventable. Additionally there is a readily available tool for measurement of peripheral nerve dysfunction.

Inasmuch as nothing has been written in the literature regarding peripheral nerve entrapment among wheelchair athletes, Chapter 2 investigates the initial questions of:

1. **Is it a problem?** What is the clinical and electrophysiological prevalence of peripheral nerve entrapments of the upper extremities among wheelchair athletes?

2. **Where is the problem?** What nerves are most commonly entrapped and what is the exact location of the entrapment?

3. **Why is there a problem?** Are there demographic or sport training/participation factors predisposing the wheelchair athlete to peripheral nerve entrapment?

With insight gained by trying to answer the questions above, Chapter 3 explores the preliminary steps of trying to intervene or treat the most common peripheral nerve entrapment syndrome in wheelchair athletes. In doing so, the following research questions are asked:

4. **Can the amount of median nerve dysfunction at the carpal tunnel resulting from a single session of heavy wheelchair propulsion be measured?** And, if so,

5. **Can this nerve dysfunction be minimized by providing external protection over the nerve during wheeling?**

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CHAPTER 2.

UPPER EXTREMITY PERIPHERAL NERVE ENTRAPMENTS AMONG WHEELCHAIR ATHLETES: PREVALENCE, LOCATION AND RISK FACTORS

INTRODUCTION

The upper extremities serve as weight bearing limbs for the wheelchair athlete. It is therefore not surprising that soft tissue injuries of the arms and hands are among the most common injuries seen in sport for athletes with a disability.[1,2] Peripheral nerve entrapments make up part of the spectrum of these soft tissue injuries. Curtis and Dillon reported that 5% of the wheelchair athletes who responded to their survey had sustained injuries characterized by hand weakness and/or numbness.[2] Similarly Martinez[3] found that 3 of 43 (7%) wheelchair road racers surveyed reported having carpal tunnel syndrome (CTS), although the diagnostic criteria for this diagnosis was not described. In a recent survey of 116 wheelchair basketball players, 22 (19%) indicated they had experienced hand numbness during the current competitive season.[4] The numbness was in an ulnar nerve distribution in 11, median nerve distribution in 7 and was nondescript in 4. It was described as occasional by 13, worse at night by 4 and exacerbated by wheeling in 2.

Although upper extremity numbness and weakness is a noted phenomenon among wheelchair athletes, no study has specifically looked at the electrophysiological prevalence, precise location, and risk factors of peripheral nerve entrapments among this growing group of sport enthusiasts.

It has however been well documented that entrapment of the median and ulnar nerves are common in various chronically disabled non-athletic populations. Prevalence rates of 10-67% by clinical and electrodiagnostic (EDX) criteria have been found among individuals with spinal cord injury[5-8], post-poliomyelitis[9] and hemiplegia[10]. Repetitive extrinsic hand pressure from use of assistive devices for mobility[5] and frequent high intracarpal pressures from wrist extension posturing[7] have both been suggested causes. The need to protect these nerves from injury has been emphasized. However, the exact location of maximal nerve injury has not been identified making placement of protective measures imprecise.

The EDX criteria for the diagnosis of CTS among the chronically disabled non-athletic groups already studied have usually been based on wrist to thenar motor latencies and wrist to digit sensory latencies.[5-7] Only one group of investigators[8,9] utilized palmar sensory techniques which allow isolation of median nerve conduction across the carpal tunnel and has been shown to increase the EDX yield by 19%.[11] Even more precise localization of nerve entrapment may be possible by applying short segment stimulation techniques of the median nerve across the carpal tunnel[12] and of the ulnar nerve across the elbow.[13,14] To date, short segment studies have not been utilized in the study of nerve entrapments among the disabled. Individuals with physical disabilities who are involved in wheelchair sport likely represent a subgroup of particularly vigorous wheelchair users who may be at increased risk of upper extremity peripheral nerve entrapment.

The purpose of this study was to:

1. Determine the prevalence of peripheral nerve entrapments of the upper extremities of wheelchair athletes by both clinical and electrophysiological criteria.

2. Localize the site of nerve entrapment as precisely as possible using short segment techniques.

3. Identify demographic and training factors associated with EDX evidence of nerve entrapment.

METHODOLOGY

Fifty two upper extremities of 28 wheelchair athletes from the Rick Hansen Centre (a fitness and training centre for people with a physical disability) and Red Deer Rebels Wheelchair Basketball Team were studied. Four arms were not studied due to previous local trauma which may have affected nerve conduction. The 8 subjects recruited from the wheelchair basketball team represented all of the disabled players on that team. The 20 subjects recruited from the Rick Hansen Centre were a "convenience" sample from a population of individuals who attended the centre and were regularly involved in wheelchair sport. Fifty-nine upper extremities of 30 age-matched, asymptomatic, able-bodied volunteers were studied as controls. Subjects with a history of a disease associated with peripheral neuropathy or with a history of injury to their hands or

arms were excluded from the study. Once the project had been explained and voluntary consent obtained, the subjects underwent a clinical examination and nerve conduction studies performed by the same physician using the same EDX machine.

The procedure was as follows:

1. Clinical examination: A clinical history was obtained from each wheelchair athlete including: age; gender; hand dominance; type and duration of disability; hours per week spent in the wheelchair; hours per week involved in wheelchair sport; presence and distribution of hand numbness, pain or weakness. A neurological examination of the upper extremities was performed including Tinel's sign and Phalen's manoeuvre, testing of motor power, pin prick sensation and inspection for wasting. The diagnostic criteria for the clinical diagnosis of CTS and ulnar neuropathy are described in Table 2-1.

2. Skin temperature: Surface skin temperature readings of the hands were taken using a plate thermistor. If the temperature was less than 32° C the arms and hands were warmed in water until that temperature was reached and then they were kept warm in a heating pad.

3. Median motor studies: With a surface recording electrode over the abductor pollicis brevis muscle, the median nerve was stimulated with a supramaximal surface impulse at the wrist (8 cm distance) and at the elbow.

4. Median sensory studies:
   a) With ring recording electrodes on the first, second, third, and fourth digits (recording electrode at PIP joint and reference electrode at DIP joint) the median nerve was stimulated at the wrist from a distance of 14 cm and the antidromic sensory nerve action potential recorded.

   b) Palmar/short segment studies: With the recording ring electrode over the PIP joint of the middle digit, antidromic sensory stimulation were applied at the palm 7 cm proximal to the PIP joint and sequentially at 2 cm intervals covering an 8 cm distance to the wrist.

A wrist stimulation at 14 cm from the recording electrode was also made and the across carpal tunnel sensory conduction velocities were calculated.

5. Radial sensory studies: With the recording ring electrodes on the thumb, the superficial radial sensory nerve was stimulated 14 cm proximally along the lateral aspect of the radius.

6. Ulnar motor conductions: With the recording electrode initially on the abductor digiti minimi (hypothenar) muscle the ulnar nerve was stimulated 8 cm proximally at the wrist. This was followed by stimulation at a point 4 cm distal to a line intersecting the medial epicondyle and the olecranon (M.E. - 0 line) with the elbow flexed to 90 degrees. These motor stimulations were repeated at 2 cm intervals proximally along the course of the ulnar nerve to a point 6 cm proximal to the M.E. - 0 line. The ulnar nerve was then stimulated near the axilla to get an upper arm conduction. The recording electrode was then moved to the first dorsal interosseous muscle (IDI) and the ulnar nerve was stimulated at the wrist location used previously.

7. Ulnar sensory conductions: With the recording electrode on the ring finger the ulnar nerve was stimulated 14 cm proximally at the wrist. Then the recording ring electrodes were switched to the fifth digit and stimulation was applied over the hypothenar eminence at the palm (7 cm), then wrist (14 cm), below elbow and finally at the above elbow position.

All EDX testing was performed using a DISA Neuromatic 2000 machine (Dantec Electronics). Filter settings were 2 Hz - 10 KHz for motor and 20 Hz - 2 KHz for sensory testing. Dantec ring sensory and disposable disc motor electrodes were used. The motor and sensory responses were recorded from the onset of the negative deflection with sensory amplitudes being measured from negative peak to positive trough, whereas the motor amplitudes were measured from baseline to negative peak. When recorded from the IDI, ulnar motor onset latencies were taken from either the initial negative or positive deflection.

In total this divided each upper extremity into 7 nerve segments: median nerve - wrist, forearm; radial nerve (sensory) - wrist; ulnar nerve - wrist, forearm; across elbow; and upper arm. The

median nerve at the wrist and ulnar nerve across the elbow segments were further subdivided into 2 cm segments.

ANALYSES

The EDX results of both upper extremities of the 30 control subjects were analyzed using means and standard deviations. From these, an abnormal result was defined as one falling more than 2 standard deviations from the mean. The number of abnormal results per nerve segment for the 52 arms of wheelchair athletes was compared with the number found in the 59 arms of the control subjects using chi-squared analysis. If a pattern of C8/T1 motor neuron or proximal motor axonal loss was seen (absent or reduced median and ulnar CMAP amplitudes with relative preservation of SNAP amplitudes and CV), the motor conduction study values were not utilized in the analysis. Significant relationships between the independent variables (age, duration of disability, time spent in the wheelchair per week and time involved in wheelchair sport per week) and the dependant variables (distal medial motor latency, median carpal tunnel sensory conduction velocity, distal ulnar motor latency to IDI and ulnar sensory forearm conduction velocity) were made using a forward step-wise multiple regression analysis.

RESULTS

1. Demographics: Types of disabilities of the wheelchair athletes included traumatic spinal cord injury in 19 (6 quadriplegia; 13 paraplegia), cerebral palsy (3), spina bifida (2), polio - lower extremity (1), multiple sclerosis (1), above knee amputation (1) and sciatic nerve injury (1). The mean duration of disability was 12 years (SD = 10.6; range = 1-12). The athletes mean age was 27.8 years (range = 16-47), whereas in the control group it was 28.5 years (range = 21-40). Twenty-five (89%) of the wheelchair athletes were male and 89% were right handed. The average time spent in the wheelchair per week was 76.5 hours (SD = 38.2; range = 6-120). The athletes average involvement in wheelchair sport was 9.8 hours per week (SD = 5.9; range = 2-25).

2. Prevalence of peripheral nerve entrapment: Five of 22 wheelchair athletes (23%) had nerve entrapment by clinical criteria (quadriplegics excluded). Four cases of CTS were evident in

3 athletes, whereas 3 cases of ulnar neuropathy were clinically present in 2 athletes. Overall, 10% of the 40 upper extremities examined had clinical CTS and 7% had ulnar neuropathy.

EDX investigation revealed a higher prevalence of peripheral nerve entrapment than was appreciated clinically. The mean and standard deviation values and EDX criteria for neuropathy are described for the median and radial nerves in Table 2-2, and for the ulnar nerve in Table 2-3. By these criteria 17 of the 28 athletes (61%) had upper extremity nerve entrapments. There were 13 cases of CTS in 9 athletes and 13 cases of ulnar neuropathy in 10 athletes. Four athletes had both CTS and ulnar neuropathy by EDX criteria.

The carpal tunnel was the most common site of nerve conduction abnormality followed by the wrist and forearm segments of the ulnar nerve (Figure 2-1). No significant abnormalities were found in the forearm segment of the median nerve, wrist segment of the radial sensory nerve or across elbow and upper arm segment of the ulnar nerve. The nerve conduction tests for CTS which were most commonly abnormal were, in descending order: distal motor latency, palmar sensory conduction velocity and median sensory conduction velocity to the ring finger. The severity of abnormality for these 3 tests in those athletes meeting EDX criteria of CTS were such that 18 were between 2 and 3 standard deviations (SD) of the mean, whereas 7 were between 3 and 4 SD of the mean. For the ulnar nerve the most commonly abnormal EDX tests were the distal motor latency to the first dorsal interosseous muscle and the ulnar sensory conduction velocity of the forearm. The abnormalities for these 2 tests were all between 2 and 3 SD except on 2 occasions, which were between 3 and 4 SD. Five of the 6 quadriplegics tested (4-C6; 1-C7; 1-C8) had EDX results suggestive of anterior horn cell or nerve root loss at the C8/T1 level.

3. Precise localization of site of nerve entrapment: The median nerve antidromic short segment sensory stimulations identified the proximal portion of the carpal tunnel to be the most prevalent site of conduction slowing (Figure 2-2). The segment 15 to 13 cm. proximal to the middle digit PIP joint was particularly affected. The ulnar nerve motor short segment conductions across the elbow identified the distal portion (4 to 2 cm. distal to the ME-0 line) to be significantly slowed.

4. Demographic factors associated with nerve entrapments: Disability duration was found to be positively associated with distal median motor latency and negatively associated with median sensory carpal tunnel conduction velocity ($p < .05$). No significant association was found between median motor and sensory conductions across the carpal tunnel and age, hours per week spent in the wheelchair, or hours per week involved in wheelchair sport. The ulnar wrist to first dorsal interosseous motor and forearm sensory conductions showed no significant association with the independent variables analyzed.

DISCUSSION

Because subject selection for this study was not randomized, the potential for selection bias exists. Athletes may have been more inclined to participate in the study because they had experienced nerve symptoms. On the other hand, athletes with nerve entrapments serious enough to preclude wheelchair sport participation would not have been included in this study. Given these limitations, the prevalence of upper extremity nerve entrapment by clinical criteria among the wheelchair athletes studied was 23%. This is in keeping with the prevalence of hand numbness, pain or weakness symptoms in competitive wheelchair basketball players[4], but is higher than the prevalence reported by other authors.[2,3] This discrepancy is likely related to the method by which the information regarding hand nerve entrapment symptoms was acquired. In this project, as well as the wheelchair basketball injury survey[4], the athletes were asked specifically about hand symptoms of numbness, weakness or pain, whereas in the other surveys the information was volunteered by the athletes when they were asked to describe the injuries they had experienced in a specific time period. The electrodiagnostic prevalence of peripheral nerve entrapment among these disabled athletes was 61%, which is in keeping with the rates reported for the chronically disabled non-athletic populations.[5-10]

The disparity between the clinical and electrodiagnostic prevalence and severity of peripheral nerve entrapments presents a treatment dilemma. On the one hand, allowing hand disability in terms of pain, weakness and numbness to occur in an individual who uses the upper extremities to compensate for other disabilities is tragic. On the other hand, unnecessary surgery, post-operative immobilization and potential complications pose remarkable additional disability for someone who is dependent on the upper extremities to perform the activities of daily living,

vocational and avocational functions. Tun and Upton describe successful carpal tunnel release in 9 chronic paraplegics with troublesome, symptomatic and electrodiagnostically proven CTS.[6] However, they also describe performing surgical releases on 3 asymptomatic hands which had significantly abnormal nerve conduction studies and which had symptomatic CTS on the contralateral side. It is the author's view that, despite the high prevalence of EDX diagnosed median nerve dysfunction at the carpal tunnel, to make the diagnosis of this "syndrome" one must have the clinical constellation of symptoms and signs. The EDX findings serve as an extension of the clinical examination.

CTS was the most common upper extremity peripheral nerve entrapment found in our series. The cause of CTS among the disabled is likely multifactorial. Previous research has documented that intracarpal tunnel pressure correlates positively with the severity of median nerve electrophysiological dysfunction.[21] High intracarpal pressures have been documented with forced wrist extension posturing[7,21,22] which is a commonly assumed position during wheeling and transferring amongst the disabled. Additionally, isometric and isotonic contraction of the finger flexors which travel within the carpal tunnel have been shown to result in increased carpal tunnel pressures 3 to 6 times resting levels.[21] Repetitive strain and overuse of the flexor tendons can also result in inflammation, swelling and tenosynovitis which may encroach on the median nerve within the carpal tunnel as well. Degenerative arthrosis of the hand and wrist has also been documented to be more among the disabled utilizing upper extremity dependent mobility aids.[23] Such arthrosis could also contribute to the development of CTS by decreasing the size of the tunnel lumen. The short segment studies in this study identified maximal median nerve dysfunction at the proximal portion of the carpal tunnel. Although the mode of measurement for the palmar short segment stimulation studies was different in this project as compared to that described in able-bodied individuals[11], it is estimated that the site identified amongst the disabled athletes is slightly more proximal. This likely corresponds to the region of hand/wheelchair interface. This supports the suggestions that repetitive extrinsic pressure of mobility aids, such as canes, walkers and wheelchairs, over the volar soft tissue covering of the carpal tunnel could contribute to the development of CTS. If this is indeed a significant contributor, it may offer an avenue of preventative or conservative treatment in terms of cushioning the hand/mobility aid interface. Further research into this area is recommended.

In our series the prevalence of ulnar neuropathy was 39% by electrophysiological criteria, and was most prevalent in the region of Guyon's canal. Although most commonly described in cyclists who apply prolonged compressive forces in this area,[24] the same mechanism may contribute to its prevalence among wheelchair athletes. As is recommended for cyclists, the use of a glove with padding over the heel of the hand may offer some protection for both the ulnar and median nerves in this region. A mild, but increased prevalence of slowing of ulnar nerve sensory conduction in the forearm segment may reflect compression at the distal aspect of the cubital tunnel (deep forearm flexor pronator aponeurosis), as this corresponded to the only site of slowing with the motor short segment stimulation studies performed. Entrapment of the ulnar nerve at this point usually occurs between the 2 heads of the flexor carpi ulnaris muscle. Heavy, repetitive contraction of this muscle, as would be required by a wheelchair athlete, prolonged elbow flexion, and repetitive extrinsic pressure in this area when the forearm rests against the wheelchair armrest may all be contributing factors.

The 3 hand muscles from which the compound motor action potentials were recorded in this study all derive their motor innervation from C8 and T1. Five of the 6 quadriplegics included in this cohort had spinal cord injuries above the C8/T1 level, and it was therefore thought that the peripheral nerve innervation to the hand musculature would thus be preserved. This, however, was not the case, suggesting more widespread spinal cord damage affecting the C8/T1 anterior horn cells (i.e. syringomyelia[25]) or nerve roots. Despite the disappearance of the motor function of the hands in these quadriplegic subjects, the lateral aspects of the hands maintain sensory input which is vitally important for them. This could be jeopardized by entrapment of the median nerve at the carpal tunnel.

This series identified a significant association between disability duration and increasing amounts of median nerve motor and sensory dysfunction, which is in keeping with some authors[5] but in contradiction to others.[8,9] This association lends support to the perspective that carpal tunnel syndrome amongst the physically disabled is likely a cumulative stress injury. It may be similar to the model of occupationally induced carpal tunnel syndrome, which has been found to be proportional to repetitiveness and, to a lesser degree, intensity of hand work.[26] As is the case in industrial medicine, strategies to minimize cumulative stress and pressure of the peripheral nerves of the upper extremities need to be identified so that preventative and conservative

measures for treatment of these prevalent, and potentially disabling peripheral nerve entrapments can be found.[27,28] In order to measure the value of such interventions, a working model of acute nerve injury with heavy wheeling would be invaluable.

TABLE 2-1: Clinical Criteria for the Diagnosis of Upper Extremity Entrapment Neuropathy

Carpal Tunnel Syndrome:

a) a history of nondescript or lateral hand numbness, and

b) clinical and examination findings of at least one of the following: positive Tinel's sign at the carpal tunnel, positive Phalen's maneuver, lateral hand hypesthesia, weakness of thumb abduction

Ulnar Neuropathy:

a) a history of nondescript or medial hand numbness and

b) clinical and examination findings of at least l of the following: positive Tinel's sign at the ulnar groove or Guyon's canal, interosseous muscle weakness, medial hand hypesthesia.

*Quadriplegics were not included in the clinical assessment.

TABLE 2-2: Control Values for Median and Radial Nerve Conductions and EDX Criteria for Entrapment Neuropathy

Nerve Stimulation - Recording Sites	Latency (ms)	Conduction Velocity (CV) (m/s)	Amplitude (μv sensory) (mV motor)	EDX Criteria for Neuropathy (>2 SD beyond control means)
Median Motor				Carpal tunnel syndrome:
· wrist to thenar	3.60 ± .33		12.4 ± 3.0	normal radial sensory conduction plus at least
· elbow to thenar		59.7 ± 4	12.2 ± 3.1	1 of the following:
				· distal motor latency > 4.3 ms
				· sensory CV to:
				digit 1 < 49 m/s
				digit 2 < 47 m/s
				digit 3 < 46 m/s
				digit 4 < 44 m/s
				wrist to palm < 42 m/s
Median Sensory				
· wrist to digit 1		60.2 ± 5.7	40.0 ± 21.4	Prolonged carpal tunnel short segments:
· wrist to digit 2		58.1 ± 5.2	46.8 ± 18.6	· 7-9 cm from PIP > .52 ms
· wrist to digit 3		55.8 ± 4.6	57.3 ± 23.1	· 9-11 cm from PIP > .61 ms
· wrist to digit 4		54.9 ± 5.0	31.4 ± 15.1	· 11-13 cm from PIP > .60 ms
· palm to digit 3		57.3 ± 4.8	50.0 ± 27.5	· 15-13 cm from PIP > .48 ms
· wrist to palm*		55.8 ± 6.5		
Median Sensory short segment studies:				Forearm median motor neuropathy
· 7-9 cm. from PIP to digit 3	.30 ± .11			if CV < 53 m/s
· 9-11 cm. from PIP to digit 3	.38 ± .11			
· 13-11 cm. from PIP to digit 3	.32 ± .12			Radial sensory neuropathy
· 15-13 cm. from PIP to digit 3	.28 ± .10			if CV < 48 m/s
Radial Sensory				
· forearm to digit 1		59.4 ± 5.6	15.5 ± 6.1	

* calculated value

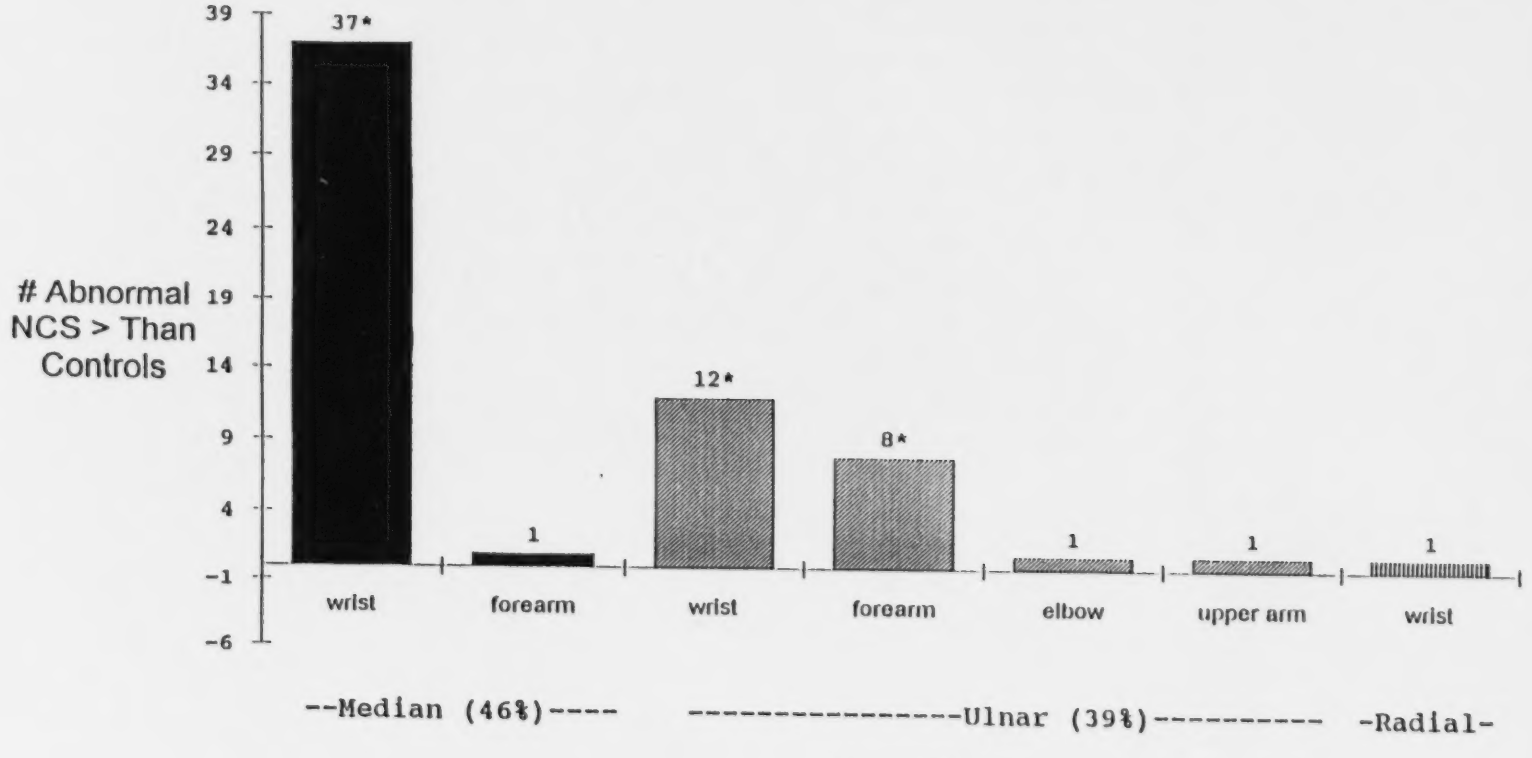
TABLE 2-3: Control Values for Ulnar Nerve Conductions and EDX Criteria for Entrapment Neuropathy

Nerve	Latency (ms)	Conduction Velocity (m/s)	Amplitude (μv sensory) (mV motor)	EDX Criteria for Neuropathy (>2 SD beyond mean)
Ulnar Motor				Wrist/hand (Guyons) segment: normal radial sensory and forearm ulnar motor conductions and at least 1 of the following: • wrist to ADM latency > 3.6 ms • wrist to IDI latency > 4.3 ms • wrist to palm sensory CV < 43 m/s • wrist to D4 CV < 46 m/s • wrist to D5 CV < 50 m/s
· wrist to hypothenar	3.1 ± .3	65.6 ± 6.0	9.7 ± 1.6	
· below elbow to hypothenar		62.7 ± 6.2	9.1 ± 1.8	
· above elbow to hypothenar		67.9 ± 5.2	9.3 ± 1.9	
· axilla to hypothenar			8.8 ± 1.7	
· wrist to IDI	3.5 ± .4		9.6 ± 3.1	
Across Elbow Motor short segments				Forearm: normal across elbow ulnar conductions plus 1 of: • motor CV < 50 m/s • sensory CV < 55 m/s
· 4-2 cm. below ME-0 line to hypothenar	.27 (.13)			
· 2-0 cm. below ME-0 line to hypothenar	.31 (.10)			
· 0-2 cm. above ME-0 line to hypothenar	.32 (.10)			
· 2-4 cm. above ME-0 line to hypothenar	.32 (.11)			
· 4-6 cm. above ME-0 line to hypothenar	.27 (.13)			
Ulnar Sensory				Across elbow: normal upper arm ulnar conductions plus 1 of: • motor CV < 46 m/s • sensory CV < 55 m/s • motor amplitude drop > 20%
· wrist to digit 4		55.7 ± 4.7	30.8 ± 12.2	
· palm to digit 5		54.6 ± 5.5	52.8 ± 24.1	
· wrist to digit 5		58.3 ± 4.2	49.0 ± 29.2	
· wrist to palm*		62.8 ± 9.5		
· below elbow to digit 5		64.6 ± 4.7	49.0 ± 29.2	
· above elbow to digit 5		65.7 ± 5.0		
				Prolonged across elbow short segments: 4-2 cm. below ME-0 line > .53 ms 2-0 cm. below ME-0 line > .51 ms 0-2 cm. above ME-0 line > .52 ms 2-4 cm. above ME-0 line > .54 ms 4-6 cm. above ME-0 line > .53 ms
				Upper arm: • motor CV < 57 m/s • sensory CV < 55 m/s • motor amplitude drop > 20%

* calculated value

FIGURE 2-1

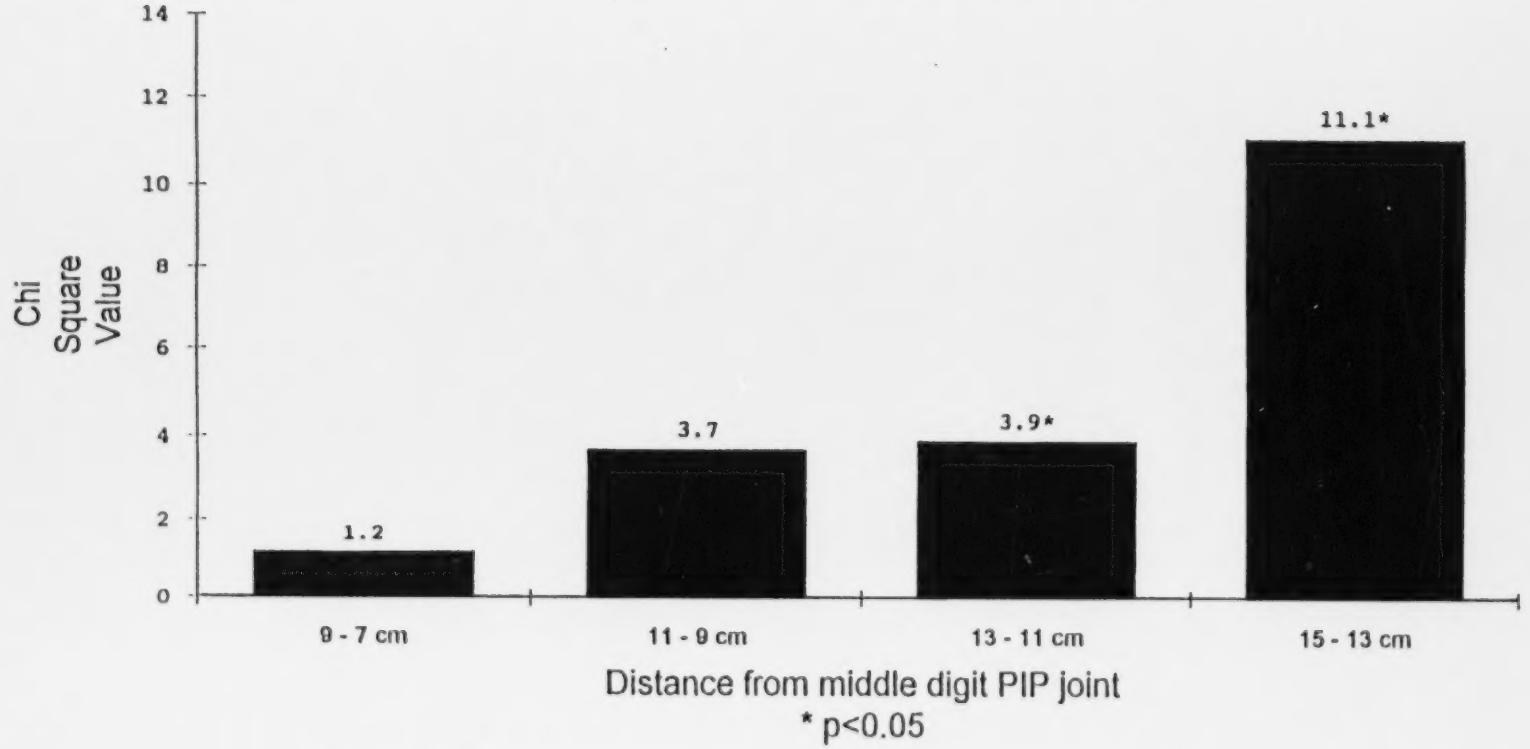
Upper extremity nerve entrapments in wheelchair athletes



* $p<0.05$

FIGURE 2-2

Abnormalities of median nerve sensory conduction within the carpal tunnel of wheelchair athletes



* $p<0.05$

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CHAPTER 3.
ACUTE MEDIAN NERVE DYSFUNCTION FROM WHEELCHAIR PROPULSION: THE DEVELOPMENT OF A MODEL AND STUDY OF THE EFFECT OF HAND PROTECTION

INTRODUCTION

Carpal tunnel syndrome (CTS) is the most commonly reported nerve entrapment syndrome.[1] Occupational factors suggested to be associated with its development include exposure to forceful and repetitive hand motion, awkward postures (including wrist extension and ulnar deviation), vibration and mechanical compression at the base of the palm.[2,3] Individuals with physical disabilities often utilize devices for mobility assistance such as walkers, canes and wheelchairs. Their hands and wrists thus become weight bearing and are required to sustain forces and postures similar to those described as occupational CTS risk factors. It is thus not surprising that the clinical and electrodiagnostic (EDX) prevalence of CTS among the disabled has been reported to be between 10 and 67%.[4-9] Wheelchair athletes put particularly heavy stresses through their hands and wrists. In previous work with 28 wheelchair athletes we found CTS prevalence to be 14% by clinical assessment and 46% by EDX criteria.[10] In addition, we also found that short segment median sensory conductions localized the site of maximal nerve conduction slowing to the proximal portion of the carpal tunnel (11 to 15 cm. proximal to the proximal interphalangeal (PIP) joint).[10] As this corresponded to the point of contact between the hand and wheel during wheelchair propulsion, it was hypothesized that repetitive extrinsic pressure over the carpal tunnel contributed to the development of CTS in wheelchair users and thus, hand protection and padding in the region of the proximal carpal tunnel might serve as a prophylactic and/or conservative treatment modality. If such protection was realized, it would be a valuable treatment adjunct since CTS, its surgery and post operative recovery period pose particularly burdensome challenges for those who rely on their hands to compensate for lower extremity disability. It would also follow that extrinsic hand protection could have application in the prevention and treatment of occupationally induced or aggravated CTS.

In order to measure the efficacy of hand protection as a strategy to prevent or treat CTS, it would be advantageous to be able to quantitate the extent of acute median nerve dysfunction resulting

from a single bout of traumatic hand use (i.e. wheeling). The development of such a measure would significantly enhance the power of future CTS investigations. To date the majority of CTS research has been epidemiologic. Because a direct measure of hand use related acute median nerve dysfunction has not been available, the cohort, case control or cross sectional survey designs which are commonly in use provide weak cause-effect inferences. As is consistent with the concept of repetitive strain injury, the assumption of an acute micro injury model is that repeated episodes of acute median nerve dysfunction accumulate to eventually result in persistent chronic median nerve dysfunction which is associated with the clinical signs and symptoms of CTS. Therefore, it would be assumed that interventions that could reduce the extent of acute median nerve dysfunction from a single session of traumatic hand use, would also reduce the probability of developing clinical CTS.

Pathophysiologically, high intracarpal pressures from traumatic hand use could cause median nerve ischemia[11] and/or paranodal demyelination[12] which would be electrodiagnostically (EDX) manifested as conduction block and conduction slowing. Nerve conduction studies which include stimulation both distal and proximal to the carpal tunnel allow these indices of median nerve injury to be measured. Using these techniques immediately before and after a session of traumatic hand use could allow the amount of median nerve conduction block and/or slowing resulting from a single session of hand trauma to be measured. In assessing the effects of traumatic hand activity (i.e. wheelchair propulsion) on nerve conduction across the carpal tunnel, several physiologic and technical factors need to be considered. The physical work associated with the traumatic hand activity can cause an increase in body temperature, and the resulting nerve warming would increase nerve conduction velocity and possibly a reduction in action potential amplitude/area.[13] Conduction block quantification, defined for purposes of this study as the percentage of waveform amplitude/area drop with stimulation distal as compared to proximal to the carpal tunnel, would not be affected by temperature alterations if nerve temperature changes affect the nerve uniformly. However, if exercise induced hand temperature increases were less pronounced distally as compared to proximally (ie. due to peripheral vasoconstriction or cooling from sweat evaporation), waveforms evoked with distal stimulation could be larger than those evoked from proximal stimulation. This could theoretically produce a "pseudo-conduction block" simply on the basis of temperature effect. An additional factor which could mimic conduction block is temporal dispersion which results in motor unit action

potential desynchronization and phase cancellation manifesting as reduced waveform amplitudes and areas.[14,15]

Given the above problem and the inherent complexities involved in its study, this investigation undertook to:

1. determine if dysfunction of the median nerve (conduction block and slowing) occurred at the carpal tunnel after a single session of vigorous exercise not involving hand trauma. Additionally, the test-retest reliability of the EDX techniques utilized was determined.

2. determine if dysfunction of the median nerve (conduction block and slowing) occurred at the carpal tunnel following a single session of vigorous wheelchair propulsion; and if so,

3. determine if the wheeling induced dysfunction could be minimized by protecting the hand using a glove with padding over the carpal tunnel; and, of secondary interest, to

4. determine if being wheelchair dependent versus non-wheelchair dependent affects the median nerve response to heavy wheeling and glove protection.

METHODOLOGY

a. Subjects - To investigate the first objective (to determine if median nerve dysfunction occurred after vigorous exercise not involving hand trauma and to document reliability) a pilot project was conducted with a convenience sample of 11 healthy, non-wheelchair dependent subjects. The exercise stimulus for this group was stationary cycling. To investigate the remaining objectives another convenience sample of 35 subjects was utilized: 16 were wheelchair dependent and 19 were non-wheelchair dependent. For these 35 subjects the exercise stimulus was wheeling. No subject had any disease entity related to peripheral neuropathy. For purposes of analysis, the wrists of each subject were treated as independent, thereby doubling the number of median nerves investigated. This approach of treating each studied wrist independently has potential shortcomings, but is at best conservative in terms of statistical power.

b. Test protocol:

i) **Pre-exercise EDX studies** - Immediately prior to the exercise stimulus, nerve conduction studies of the motor and sensory components of both median nerves were performed. All EDX testing was performed by the same investigator using the same electrodes and EDX machine (Neuroport 1 - Biotron Inc.). Disposable Dantec (13L20) surface electrodes were used for motor conductions. The filter settings were 2 Hz - 10 KHz for motor and 20 Hz - 2 KHz for sensory testing. Testing sequence remained consistent for all subjects, with the left hand being tested first followed by the right hand.

a) ***sensory***: the sensory conductions were obtained by antidromic surface stimulation at both the wrist and palm. Sensory nerve action potentials were recorded using Dantec (13L69) ring electrodes on the middle digit positioned with the reference electrode around the distal interphalangeal (DIP) joint and the recording electrode around the PIP joint. The stimulus was placed over the palm 7 cm proximal to the PIP joint and at the wrist 14 cm proximal to the PIP joint. A stimulus of 0.1 msec was used. The stimulus intensity was gradually increased until a supramaximal recording was obtained. Care was taken to use the minimal stimulus intensity necessary to obtain a maximal amplitude wave form.

b) ***motor***: the median nerve motor conductions across the carpal tunnel were recorded using an active surface electrode over the motor point of the abductor pollicis brevis muscle. The reference electrode was placed over the metacarpal phalangeal (MCP) joint of the thumb. The cathode of the stimulator was placed on the palm at a point where the tip of the flexed fourth digit contacts the palm, as described by Pease et al.[16] The median nerve was then stimulated at the wrists 8 cm. proximal to the recording electrode. Each motor and sensory waveform was averaged 5 times. The sites of the recording and stimulating electrodes were marked with indelible ink so the same location could be used for the post-exercise nerve conduction testing in order to maximize consistency of electrode placement. The parameters of conduction velocity (recorded from the onset of the negative deflection), amplitude (from base-line to negative peak) and integrated area under the negative wave were

recorded. Surface skin temperature recordings were obtained over the proximal portion of the carpal tunnel using a plate skin thermistor. These recordings were made at the time of the motor and sensory conductions for both the pre and post exercise testing periods. The EDX parameters of interest were the across carpal tunnel conduction velocities (motor and sensory) and the percentage of conduction block (motor and sensory).

Across carpal tunnel motor and sensory conduction velocities were calculated as:

$$\frac{DCT}{LCT}$$

where: DCT = the distance across the carpal tunnel as measured by the distance between the palm and wrist stimulation points (7 cm).

LCT = the latency of conduction across the carpal tunnel which was calculated by subtracting the palm to digit (sensory) or palm to abductor pollucis brevis (motor) latency from the respective latencies obtained from wrist stimulation.

The percentage of conduction block of the median motor and sensory fibres was calculated as:

$$\frac{P - W}{P} \times 100$$

where: P = the amplitude (area) of the negative wave evoked by stimulation at the palm

W = the amplitude (area) of the negative wave evoked by stimulation at the wrist

As an index of waveform temporal dispersion, the latency between the negative wave onset and peak (rise time) was calculated for both compound motor and sensory action potentials recorded from wrist stimulation pre and post exercise.

Following the pre-exercise EDX testing of the 11 pilot subjects, the electrodes were removed and then reapplied to the left hand, and the same testing protocol was repeated so EDX test-retest reliability could be calculated.

ii) **Exercise stimulus**

a) Following the pre-exercise EDX studies, the 11 subjects in the pilot group rode a stationary exercise bike for 30 minutes. They were instructed to keep their hands on their laps or at their sides, and not to lean their hands against or grip the bike handle bars. They were allowed to choose their own cycling resistance and speed, but were instructed to exercise at the maximal intensity they could tolerate for 30 minutes.

b) The 35 subjects in the study group propelled a sport wheelchair which was mounted on a stationary wheelchair ergometer. The wheelchair was designed for use in wheelchair basketball. On one hand they wore a synthetic leather cycling glove which had ¼ inch foam padding sewn into the region covering approximately the proximal half of the carpal tunnel. The hand that was assigned the glove was randomized by coin toss. The mounted wheelchairs were propelled for 2,000 hand strikes at a maximal self-selected cadence. This cadence was established by the subject at the beginning of the exercise session and then maintained throughout the session by keeping time with a metronome.

iii) **Post-exercise EDX testing** - Immediately post-exercise, the same nerve conduction and temperature protocol, as described in (i) was repeated on each hand.

ANALYSES

For the pilot group, test-retest values were analyzed for differences using a dependent T-test and for correlation using a Pearson correlation coefficient. To analyze across carpal tunnel conduction velocity a 3-way (with 1 factor repeated) Analysis of Covariance model was utilized where time (pre, post exercise) was the repeated measure and hand protection and wheelchair dependency were the remaining 2 independent factors. The hand temperature measures taken at each EDX testing session were used as the covariate. To analyze conduction block changes, a similar statistical model to the above was used, except the covariate of temperature was eliminated, as the impact of temperature change on conduction block was theoretically not

needed. Pre and post exercise hand temperatures and waveform rise times were compared with dependent t-tests. For all statistical tests of significance a 2-tailed level of significance at .05 was used.

RESULTS

a. Pilot Project - Eleven healthy, non-wheelchair dependent subjects participated in the pilot project. Their mean age was 33.5 years (SD = 7.4). The means, standard deviations and correlations of all pre-cycle test-retest EDX testing for the pilot group subjects are summarized in Table 3-1. No significant differences were found between any motor or any sensory test-retest means. All were found to correlate significantly except measures of sensory conduction block (amplitude and area).

   Table 3-2 summarizes the data reflecting the effect of exercise, devoid of hand trauma, on hand temperature and median nerve conduction. While there was a statistically significant rise in surface hand temperature, there were no significant changes in across carpal tunnel motor/sensory conduction velocity or motor/sensory conduction block.

b. Median Nerve Dysfunction with Wheeling - The average age and wheeling cadence of the 35 subjects participating in this part of the investigation are presented in Table 3-3. No statistically significant difference was found for these parameters between the wheelchair dependent and independent subgroups. Hand surface temperature rose significantly with wheeling. Across carpal tunnel motor or sensory conduction velocities were not significantly affected by the hand trauma of wheeling. However, there were significant increases in the percentage of median motor amplitude and area conduction block, as well as the percentage of sensory amplitude conduction block following vigorous wheelchair propulsion (Table 3-4). There was no evidence of waveform temporal dispersion with wheeling in terms of prolongation of waveform rise times. A comparison of the mean net across carpal tunnel conduction block changes (conduction block post-exercise minus conduction block pre-exercise) following cycle and wheeling exercise is represented in Figure 3-1.

c. Effect of Glove Protection on Wheeling Induced Median Nerve Dysfunction - Glove protection of the hand did not significantly alter across carpal tunnel median nerve conduction velocity or conduction block changes resultant from wheelchair propulsion.

d. Effect of Being Wheelchair Dependent versus Non-Wheelchair Dependent - Median nerve conduction (pre-wheeling values) across the carpal tunnel was generally slower in the wheelchair dependent group (Table 3-5). When expressed as conduction abnormalities meeting the EDX criteria of CTS (distal motor latency > 4.3 msec and across carpal tunnel sensory conduction velocity < 43 m/sec) 7 of 32 (22%) of hands of wheelchair dependent subjects had CTS, whereas only 1 of 38 (3%) of wheelchair independent subjects' hands was affected ($p < .05$). Median nerve dysfunction from wheeling was not affected by whether or not the median nerve had EDX abnormalities compatible with CTS prior to the wheeling. Likewise, wheelchair dependency did not affect the amount of median nerve dysfunction resultant from a single session of vigorous wheelchair propulsion nor the effect of wearing a protective glove.

DISCUSSION

This study has defined a quantitative method of measuring median nerve dysfunction across the carpal tunnel resulting from a single session of heavy/repetitive hand use -- in this case wheelchair propulsion. We have called the distal to proximal carpal tunnel drop in motor and sensory area "conduction block", although it is appreciated that the definition, cause and significance of conduction block is a point of debate in the literature.[14,15] A surprising amount of "conduction block" was found at the pre-exercise testing of both the cycle and wheeling groups. This is difficult to explain in pathologic terms and may be technical in nature. However, the same technical factors would have been present at both the pre and post cycling EDX testing and statistically significant increases in "conduction block" were recorded after wheeling. Thus, it is felt the quantified measure of median nerve dysfunction is legitimate.

The conduction block identified following wheeling did not appear to be a differential hand temperature effect, as it was not seen with vigorous non-hand trauma (cycling), despite a similar hand surface temperature rise being documented. There was also no evidence of waveform

temporal dispersion (rise time increase) to account for the post-exercise conduction block. The fact that conduction block and not conduction slowing occurred with wheeling suggests that the pathophysiological insult to the nerve is ischemic in origin and may represent the "rapidly reversible physiological block" described by Gilliatt.[17] This is in keeping with the findings of Lundborg et al[11] who experimentally induced sensory and motor conduction block of the median nerve with localized pressure over the carpal tunnel. They found that the conduction block was subtle and variable with 30 mmHg intracarpal pressure, however, with increases to 60-90 mmHg it was predictable and of rapid onset. The documented conduction block persisted with supersystolic arterial occlusion at the proximal arm, despite release of the pressure over the carpal tunnel, further attesting to its ischemic origin. They found motor and sensory conduction velocities across the carpal tunnel to be minimally affected by the extrinsic compression. Interestingly, the conduction block they documented resolved within 5 to 10 minutes following release of the carpal tunnel pressure and/or tourniquet. Completion of the post-wheeling bilateral EDX studies in our project took approximately 20-25 minutes. This suggests that some of the temporary conduction block may have cleared and the values found may have actually underestimated the true extent of the block. Lundborg et al[11] found the sensory conduction block to be earlier and more predictable. Conversely, this investigation found significant conduction block of the motor amplitude and areas, whereas only the sensory amplitudes were significantly affected. This may in part be explained by the greater variability of sensory amplitude/area recordings, as is evidenced by the poor test-retest correlations found in the pilot project. It is presumed this is due to the increased amount of baseline instability encountered with sensory studies. Alternatively, the median nerve topography may be important. It has been reported that predominantly motor fibres are located on the palmar-radial aspect of the nerve.[11] These fascicles may be selectively traumatized during wheeling using the palmar grip technique.

It is suggested that, in the early stages of nerve compression, nerve ischemia is the major insult.[11] Later, as ischemia and mechanical pressure worsen, mechanical deformation with paranodal demyelination and axonal loss occurs.[12] The ability of the model described in this research project to document subtle conduction block changes may allow early identification of high CTS risk activities or individuals in order that preventative or early conservative treatment modalities can be instituted.[18] Likewise, the efficacy of such treatment could be objectively and rapidly assessed using the same model.

The need for effective preventative treatment and early intervention among the wheelchair dependent is highlighted by the high prevalence of median nerve conduction abnormalities found among the wheelchair dependent subgroup of this study.

Few reports of "dynamic" provocative tests of median nerve function at the wrist could be found in the literature. There are reports of the effect of the electrophysiological effects of sustained static posturing in positions of extreme flexion and extension.[19,20] In these investigations, distal motor and sensory latencies were the parameters of interest and only slight changes were noted, causing one investigator to conclude that the procedure did not warrant addition to the conventional EDX screening procedure for CTS.[20] A "dynamic" provocative procedure was described to enhance the EDX diagnosis of pronator syndrome.[21] This helped clarify the diagnosis in only one case, and therefore was not deemed helpful. A possible explanation for the reported difficulty in measuring EDX changes secondary to dynamic nerve compression could include: failure to account for temperature changes with exercise, failure to look at amplitude/area instead of conduction velocity changes, and excessive variability in the parameters used to measure the dependent variables of interest.

Hand protection using a glove with foam padding over the region of the carpal tunnel was ineffective in protecting the median nerve from wheeling trauma. It is possible that this type of protection was inadequate, or alternatively that extrinsic compression over the carpal tunnel is not the major cause of median nerve injury. Several studies have documented elevated carpal tunnel pressures, above levels known to result in nerve damage, related to extremes of wrist posture.[22-24] The mechanics of wheelchair propulsion require repetitive wrist posturing into extreme extension and ulnar deviation. Therefore, future investigations into modes of median nerve protection may include alternate forms of carpal tunnel padding, or devices which support the wrist and prevent extreme extension/ulnar deviation posturing.

TABLE 3-1: Median Nerve EDX Test-Retest Means (SD) and Correlations: Pilot Project (n = 11)

	MEANS (SD)				CORRELATION
	Test		Retest		
MOTOR:					
palm to thenar latency (ms)	1.7	(.3)	1.8	(.32)	.88*
palm to thenar amplitude (mv)	14.1	(3.7)	14.3	(4.0)	.83*
palm to thenar area (mvms)	44.6	(11.0)	43.5	(11.3)	.64*
wrist to thenar latency (ms)	3.5	(.4)	3.5	(.4)	.75*
wrist to thenar amplitude (mv)	12.4	(4.0)	12.2	(4.5)	.95*
wrist to thenar area (mvms)	38.2	(7.4)	36.2	(11.4)	.98*
carpal tunnel conduction velocity (m/s)	43.6	(6.0)	44.6	(6.2)	.60*
conduction block - amplitude (%)	11.8	(15.7)	14.8	(18.5)	.79*
conduction block - area (%)	16.9	(15.3)	19.0	(19.4)	.74*
SENSORY:					
palm to D3 conduction velocity (m/s)	58.1	(7.4)	58.5	(7)	.88*
palm to D3 amplitude (mv)	0.049	(.018)	0.047	(.015)	.86*
palm to D3 area (mvms)	0.030	(.009)	0.029	(.010)	.75*
wrist to D3 amplitude (mv)	0.036	(.016)	0.035	(.012)	.88*
wrist to D3 area (mvms)	0.027	(.01)	0.026	(.009)	.87*
carpal tunnel conduction velocity (m/s)	49.7	(5.4)	48.5	(4.5)	.68*
conduction block - amplitude (%)	28.6	(7.4)	26.5	(8.1)	.42
conduction block - area (%)	8.9	(12.1)	10.0	(9.9)	.02

* $p < .05$

TABLE 3-2: Effect of Cycling on Hand Temperature and Nerve Conduction: Pilot Project (n = 22)

	PRE-CYCLING Mean	SD	POST-CYCLING Mean	SD	p VALUE
Skin temperature (°C)	34.8	(1.1)	36.7	(.85)	< .0001
Across carpal tunnel:					
• motor conduction velocity (m/sec)*	42.0	(6.5)	47.5	(7.7)	.71
• sensory conduction velocity (m/sec)*	50.5	(3.9)	54.2	(6.1)	.89
• motor conduction block - amplitude (%)	18.6	(20.2)	16.1	(23.1)	.49
• motor conduction block - area (%)	19.4	(19.2)	20.5	(20.7)	.77
• sensory conduction block - amplitude (%)	26.0	(7.0)	27.7	(7.8)	.19
• sensory conduction block - area (%)	13.4	(7.0)	17.7	(10.7)	.14

* mean unadjusted for temperature difference

TABLE 3-3: Average Age and Cadence of Wheeling Group Subjects

	NUMBER	AGE (years)		WHEELING CADENCE (hand strikes/min)	
		Mean	SD	Mean	SD
Wheelchair dependent	16	34.4	(15.7)	65.5	(12.3)
Wheelchair independent	19	28.4	(7.3)	63.2	(5.9)
TOTAL:	35	31	(12.1)	64.2	(9.2)

Table 3-4: Effect of Wheeling on Hand Temperature and Nerve Conduction (n = 70)

	PRE-WHEELING Mean	SD	POST-WHEELING Mean	SD	p VALUE
Skin temperature (°C)	34.8	(1.1)	36.4	(.9)	< .0001
Across carpal tunnel:					
• motor conduction velocity (m/sec)*	41.2	(10.8)	44.5	(12.0)	.06
• sensory conduction velocity (m/sec)*	48.5	(9.0)	51.7	(8.2)	.84
• motor conduction block - amplitude (%)	6.8	(39.5)	14.5	(26.3)	.02
• motor conduction block - area (%)	11.4	(37.1)	21.4	(27.0)	.004
• sensory conduction block - amplitude (%)	19.4	(20.5)	24.6	(15.8)	.02
• sensory conduction block - area (%)	3.4	(40.0)	9.7	(24.8)	.05

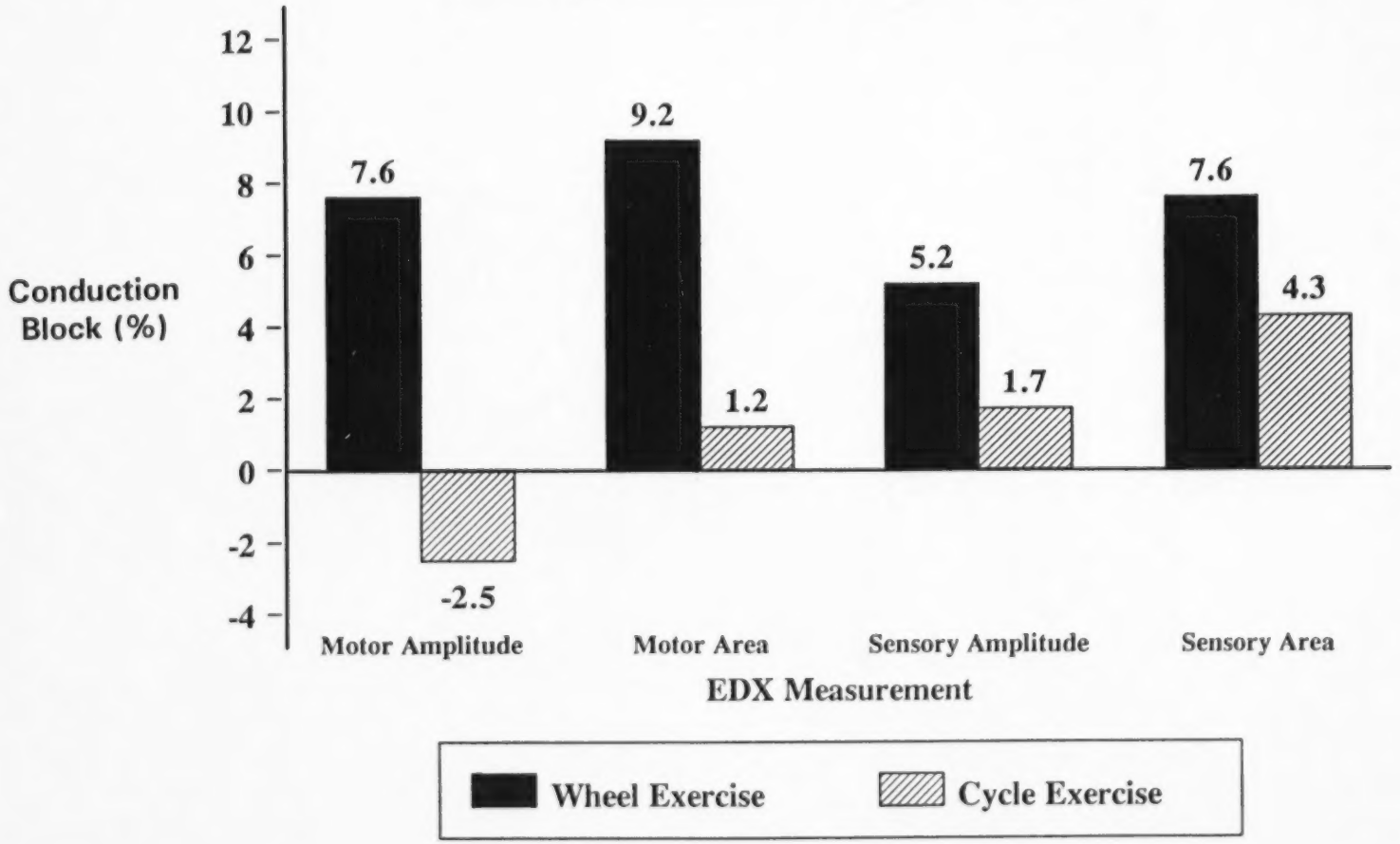
* mean unadjusted for temperature difference

Table 3-5: Median Nerve Conduction Comparison Between Wheelchair Dependent and Non-Wheelchair Dependent Subjects

Median Nerve Conduction	Wheelchair Dependent n = 32 hands Mean	SD	Non-Wheelchair Dependent n = 38 hands Mean	SD	p Value
Distal motor latency (msec)	4.1	(.7)	3.6	(.6)	.002
Across carpal tunnel motor conduction velocity (m/sec)	38.7	(10.3)	43.3	(10.7)	.07
Across carpal tunnel sensory conduction velocity (m/sec)	46.1	(8.5)	50.5	(9.0)	.04

FIGURE 3-1

Net Across Carpal Tunnel Conduction Block with Cycle and Wheeling Exercise



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CHAPTER 4.
CONCLUSIONS

The purpose of the research contained in this thesis was to investigate peripheral nerve entrapments of the upper extremity of wheelchair athletes. As stated in the introduction, 5 specific research questions were being asked. These questions and the conclusions drawn, based on the research performed, are as follows:

1. **Is there a problem?**

   Peripheral nerve entrapments of the upper extremities of wheelchair athletes are prevalent. Based on clinical criteria the prevalence is 23%, whereas the electrodiagnostic criteria based prevalence was 61%.

2. **Where is the problem?**

   The most common site of nerve entrapment, based on EDX testing, is the median nerve at the carpal tunnel - 46% prevalence. The site of the median nerve within the carpal tunnel most commonly affected is the proximal portion (11-15 cm. proximal to PIP joint of long finger). Ulnar neuropathy is the second most common entrapment electrophysiologically (39%), and occurs at the wrist (Guyon's canal) and the forearm segments (probably near the cubital tunnel).

3. **Why is there a problem?**

   Impaired median nerve function is significantly related to disability duration. No other demographic or training factors are associated with peripheral nerve entrapment.

4. **Can the amount of median nerve dysfunction at the carpal tunnel resulting from a single session of heavy wheelchair propulsion be measured?**

A statistically significant amount of conduction block can be measured following a single session of vigorous wheelchair propulsion. This pattern of median nerve dysfunction is not seen following exercise which does not involve repetitive hand trauma.

5. **Can this dysfunction be minimized by using extrinsic nerve protection?**

Protection of the median nerve using a glove with extra padding in the region of the proximal carpal tunnel does not result in reduction of the median nerve dysfunction resulting from wheeling.

Future research should utilize the model of acute nerve injury from wheeling developed in this thesis. The model could be utilized to investigate other treatment interventions aimed at protecting the median nerve at the carpal tunnel such as: alternate types of glove protection, splints which would limit the extent of wrist movement during wheeling, a combination of both the wrist splint and glove, altering the surface of the push rim or tire, or altering wheeling technique. Additionally, the model of acute nerve dysfunction secondary to wheeling needs to be investigated with respect to the problem of ulnar nerve entrapment. Similar research measuring both waveform size drop and conduction slowing of the ulnar nerve across the 2 segments identified as being common sites of entrapment (Guyon's canal and distal elbow) could be done to see if this model works for the ulnar nerve as it did for the median. If so, then ulnar nerve treatment strategies and measurements of efficacy could be measured. This model of acute nerve injury may also have application to industry where work related CTS is a common repetitive strain injury in workers who do heavy and repetitive hand activities.